

Reduced 10-Year Risk of Coronary Heart Disease in Patients Who Participated in a Community-Based Diabetes Prevention Program

The DEPLOY pilot study

ELAINE R. LIPSCOMB, PHD¹
EMILY A. FINCH, MA²
EDWARD BRIZENDINE, MS²

CHANDAN K. SAHA, PHD²
LAURA M. HAYS, RN, PHD³
RONALD T. ACKERMANN, MD, MPH²

OBJECTIVE — We evaluated whether participation in a community-based group diabetes prevention program might lead to relative changes in composite 10-year coronary heart disease (CHD) risk for overweight adults with abnormal glucose metabolism.

RESEARCH DESIGN AND METHODS — We used the UK Prospective Diabetes Study engine to estimate CHD risk for group-lifestyle and brief counseling (control) groups. Between-group risk changes after 4 and 12 months were compared using ANCOVA.

RESULTS — Baseline 10-year risk was similar between treatment groups ($P = 0.667$). At 4 and 12 months, the intervention group experienced significant decreases in 10-year risk from baseline (-3.28% , $P < 0.001$; and -2.23% , $P = 0.037$) compared with control subjects (-0.78% , $P = 0.339$; and $+1.88\%$, $P = 0.073$). Between-group differences were statistically significant and increased from the 4- to 12-month visits.

CONCLUSIONS — Community-based delivery of the Diabetes Prevention Program lifestyle intervention could be a promising strategy to prevent both CHD and type 2 diabetes in adults with pre-diabetes.

Diabetes Care 32:394–396, 2009

Adults with pre-diabetes have increased cardiometabolic risk, and intensive lifestyle interventions have been shown to improve their risk factor control (1–3). In this article, we evaluate the hypothesis that the Diabetes Education & Prevention with a Lifestyle Intervention Offered at the YMCA (DEPLOY) pilot study reduces the composite 10-year coronary heart disease (CHD) risk in overweight adults with abnormal glucose metabolism.

RESEARCH DESIGN AND METHODS — The DEPLOY study's design, participant characteristics, and results have been published separately (4). Briefly, of 131 individuals who met screening criteria at two Greater Indianapolis YMCA facilities, 92 interested adults enrolled. All participants had an elevated BMI ($\geq 24 \text{ kg/m}^2$), an American Diabetes Association risk questionnaire score of 10 or higher (5), and an abnormal whole-blood glucose concentration determined by a finger stick (110–199

mg/dl or 100–199 mg/dl if fasting ≥ 9 h). Exclusion criteria included an existing diagnosis of diabetes or any comorbidity that could limit lifespan to < 3 years or contraindicate light-moderate physical activity. Before implementation, one of the two matched YMCA sites was randomly assigned to receive training and support for delivering a formal, group-based adaptation of the Diabetes Prevention Program (DPP) lifestyle intervention. The control site only provided information about existing YMCA wellness programs.

Indiana University School of Medicine research staff measured body weight, systolic blood pressure (SBP), A1C, total cholesterol, and HDL cholesterol at baseline and at 4–6 and 12–14 months after enrollment. A One-Touch Ultra handheld glucose meter was used to determine the whole-blood glucose concentration (6), and A1C was assessed from a finger-stick capillary blood sample using a DCA 2000 analyzer (7,8). Total and HDL cholesterol were measured from capillary blood using an LDX lipid analyzer (9,10). While seated and relaxed for at least 5 min, participants' SBP was assessed once at each visit with an aneroid sphygmomanometer. The Indiana University/Purdue University Indianapolis Institutional Review Board approved the study.

Ten-year risk of CHD was predicted using the UK Prospective Diabetes Study (UKPDS) risk engine and setting diabetes duration to zero. The model is based on CHD events from UKPDS participants with type 2 diabetes; it incorporates A1C, age at diagnosis, time since diagnosis, sex, smoking, SBP, total cholesterol-to-HDL cholesterol ratio, and race (11). Although the UKPDS engine has not yet been validated for risk prediction in pre-diabetes, it is the only nonproprietary engine we are aware of that incorporates blood pressure, cholesterol, and A1C. Moreover, A1C has been shown to predict cardiovascular disease risk even at the lowest range

From the ¹Roudebush Veterans Affairs Medical Center, Health Services Research & Development Center of Excellence for Implementing Evidence-Based Practice, Indianapolis, Indiana; the ²Department of Medicine, Indiana University School of Medicine, Indianapolis, Indiana; and the ³School of Nursing, Indiana University, Indianapolis, Indiana.

Corresponding author: Elaine R. Lipscomb, elaine.lipscomb@va.gov.

Received 3 September and accepted 15 December 2008.

Published ahead of print at <http://care.diabetesjournals.org> on 23 December 2008. DOI: 10.2337/dc08-1622.

© 2009 by the American Diabetes Association. Readers may use this article as long as the work is properly cited, the use is educational and not for profit, and the work is not altered. See <http://creativecommons.org/licenses/by-nc-nd/3.0/> for details.

The costs of publication of this article were defrayed in part by the payment of page charges. This article must therefore be hereby marked "advertisement" in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

Table 1—Changes in predicted 10-year CHD risk in DEPLOY participants

	Community-based DPP				Control				Difference		
	n	Mean ± SE*	95% CI	P†	n	Mean ± SE	95% CI	P†	Mean	95% CI	P‡
4 months	39	-3.28 ± 0.76	-4.79 to -1.77	<0.001	34	-0.78 ± 0.81	-2.40 to 0.84	0.339	-2.50	-4.73 to -0.28	0.028
12 months	29	-2.23 ± 1.05	-4.33 to -0.14	0.037	30	1.88 ± 1.03	-0.18 to 3.94	0.073	-4.11	-7.06 to -1.17	0.007

n = 46 for each group at baseline. At the 4- and 12-month follow-ups, the completion rates were 85 and 83%, respectively, for the intervention group and 63 and 72%, respectively, for the control group. However, for the control group, sufficient data to construct UKPDS risk scores were not available for four additional participants at 4 months and three additional participants at 12 months. *Estimates were adjusted for baseline CHD risk. †P values for change from baseline. ‡P values for difference in change between groups.

of values among patients with diabetes (11,12). We chose this approach to maximize sensitivity for estimating the relative impact of a lifestyle intervention on changes in all three risk factors combined, not to provide absolute risk estimates.

Intervention and control group baseline CHD risk scores were compared using Student's *t* test. Change in CHD risk was calculated as 4- or 12-month risk minus baseline risk. Between-group risk changes were compared using ANCOVA with the baseline CHD risk as a covariate. Analyses were performed using SAS version 9.1 (SAS Institute, Cary, NC).

RESULTS— The mean baseline 10-year risks for intervention and control groups were similar (11.9% [95% CI 9.3–14.6] and 11.1% [8.2–14.0], respectively; *P* = 0.667). At 4 and 12 months (Table 1), the intervention group experienced significant decreases in 10-year risk from baseline; between 4 and 12 months, the CHD risk increased, but not back to baseline levels. After 4 months, the control group experienced an insignificant decrease in predicted 10-year CHD risk; at 12 months, mean CHD risk increased ~2% over baseline (*P* = 0.073). Compared with the control group, the intervention group experienced greater reductions in predicted CHD risk at each follow-up visit. Between-group differences increased from the 4- to 12-month follow-up visits.

Secondary analyses of individual risk factor changes showed either no change or minimal improvement in A1C and SBP at 4 and 12 months for both groups. However, the total cholesterol-to-HDL cholesterol ratio decreased significantly in the intervention group, whereas control subjects showed either no change or significant increase, suggesting that changes in the total cholesterol-to-HDL cholesterol ratio explained much of the between-group difference in predicted CHD risk.

CONCLUSIONS— In the DEPLOY study, a DPP lifestyle intervention deliv-

ered in YMCA facilities by centrally trained lay group instructors had a statistically significant and clinically meaningful impact on composite cardiometabolic risk as estimated by the UKPDS risk engine. This is consistent with effects of the original DPP lifestyle intervention, i.e., improved blood pressure, triglycerides, LDL cholesterol, fasting plasma glucose, and HDL cholesterol levels (13).

Although the UKPDS risk engine is specific to people with type 2 diabetes, baseline CHD risk estimates for our study population were similar to those of newly diagnosed participants in the UKPDS study (11). Compared with the UKPDS sample, DEPLOY participants were on average ~6 years older and more often female (56 vs. 42%), with lower A1C (5.6 vs. 6.7 mg/dl), comparable SBP, lower total cholesterol (188 vs. 209 mg/dl), and higher HDL cholesterol (46 vs. 43 mg/dl).

This was a small study with notable limitations. First, we used the diabetes-specific UKPDS risk engine to predict relative probabilities for CHD in persons with pre-diabetes. Because we set duration of diabetes to zero and it may take several years for diabetes to develop, the predicted risk may be overestimated. However, intervention and control groups were enrolled using identical criteria; thus, any possible overestimate of risk should occur similarly in both groups and should not affect comparisons. Second, at 12 months there was an approximate 33% overall dropout rate; however, it was similar between groups. Furthermore, baseline 10-year CHD risk and all risk factors included in the CHD risk calculation were similar for dropouts and nondropouts in each group. Third, the control group experienced a greater and more rapid increase in CHD risk between 4 and 12 months mediated largely by increases in the total cholesterol-to-HDL cholesterol ratio compared with the intervention group, contributing significantly to the between-group difference.

In conclusion, significant reductions in estimated 10-year CHD risk suggest that this model for community-based delivery of the DPP lifestyle intervention could have a significant effect on prevention of both CHD and type 2 diabetes in overweight adults with abnormal glucose metabolism.

Acknowledgments— Support for this study was provided by the National Institute of Diabetes and Digestive and Kidney Diseases (R34 DK70702-02) and Indiana University School of Medicine.

No potential conflicts of interest relevant to this article were reported.

We recognize the support and participation of the YMCA of Greater Indianapolis and the involvement of all DEPLOY study participants.

References

1. Benjamin SM, Valdez R, Geiss LS, Rolka DB, Narayan KM: Estimated number of adults with prediabetes in the U.S. in 2000: opportunities for prevention. *Diabetes Care* 26:645–649, 2003
2. Haffner SM: Pre-diabetes, insulin resistance, inflammation and CVD risk. *Diabetes Res Clin Pract* 61 (Suppl. 1):S9–S18, 2003
3. Leiter LA: From hyperglycemia to the risk of cardiovascular disease. *Rev Cardiovasc Med* 7 (Suppl. 2):S3–S9, 2006
4. Ackermann RT, Finch EA, Brizendine E, Zhou H, Marrero DG: Translating the Diabetes Prevention Program into the community: the DEPLOY pilot study. *Am J Prev Med* 35:357–363, 2008
5. American Diabetes Association: Diabetes dropout Risk Calculator [Internet], 2008. Available from <http://www.diabetes.org/risk-test.jsp>. Accessed 7 November 2008
6. American Diabetes Association: Screening for type 2 diabetes. *Diabetes Care* 26 (Suppl. 1):S21–S24, 2003
7. Marrero DG, Vandagriff JL, Gibson R, Fineberg SE, Fineberg NS, Hiar CE, Crowley LE: Immediate HbA1c results: performance of new HbA1c system in pediatric outpatient population. *Diabetes Care* 15:1045–1049, 1992
8. John WG, Edwards R, Price CP: Labora-

- tory evaluation of the DCA 2000 clinic HbA1c immunoassay analyser. *Ann Clin Biochem* 31:367–370, 1994
9. Bard RL, Kaminsky LA, Whaley MH, Zajakowski S: Evaluation of lipid profile measurements obtained from the Cholestech L.D.X analyzer. *J Cardiopulm Rehabil* 17: 413–418, 1997
 10. Santee J: Accuracy and precision of the Cholestech LDX system in monitoring blood lipid levels. *Am J Health Syst Pharm* 59:1774–1779, 2002
 11. Stevens RJ, Kothari V, Adler AI, Stratton IM: The UKPDS risk engine: a model for the risk of coronary heart disease in Type II diabetes (UKPDS 56). *Clin Sci (Lond)* 101:671–679, 2001 [erratum in *Clin Sci (Lond)* 102:679, 2002]
 12. Khaw KT, Wareham N, Luben R, Bingham S, Oakes S, Welch A, Day N: Glycated haemoglobin, diabetes, and mortality in men in Norfolk cohort of European prospective investigation of cancer and nutrition (EPIC-Norfolk). *BMJ* 322:15–18, 2001
 13. The Diabetes Prevention Program Research Group: Impact of intensive lifestyle and metformin therapy on cardiovascular disease risk factors in the diabetes prevention program. *Diabetes Care* 28:888–894, 2005